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ORIGINAL ARTICLES

DIAGNOSIS OF GASTROINTESTINAL DISEASE.*

Illustrated by Forty Operative Cases—April-October, 1914.

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Before I begin my discussion I wish to state that these are forty consecutive operatively confirmed diagnoses out of the 170 gastrointestinal cases which I have seen in the last six months. I purposely confine myself to these few cases which I have seen in this city as I wish to show that we can diagnose as well here as in Rochester or Europe. I present operatively confirmed diagnoses because I hope that after hearing about these cases you will believe that the same percentage of correctness holds good in my non-operative cases. My paper will not attempt to discuss all my cases because I would have to cover a greater part of the field of gastroenterology. I will merely discuss a few interesting points in this report of forty cases.

Schmieden in the Grenzgebieten last March* made correct diagnoses in about 85% of his cases. This he did within a reasonable time after the patient had entered the hospital. He did not dawdle until the patient was moribund and beyond surgical aid. Turning a patient over to the surgeon at this late stage is a vicious and horrible practice. I speak of this particularly because of the large percentage of poor operative results reported by some of my fellow internists in this and other cities.

In discussing Schmieden's paper at the time I maintained that we could arrive at practically the same percentage of correctness in non-acute gastrointestinal disease if we did three things thoroughly: firstly, make a good history; secondly, make a thorough fluoroscopic examination; thirdly, examine the stool for occult blood or with the Schmidt test diet as the case requires. I rate these in the order of their importance. All other examinations are of lesser importance and contribute other than confirmatory evidence only in exceptional cases. Of course, I am talking of diseases which lie beyond the reach of the proctoscope. There is no single gastrointestinal case in my operative series which could have been better diagnosed if other methods of diagnosis had been employed.

About history taking we all know, the value of

the Schmidt test diet I hope to take up in a later paper. To-night I shall discuss chiefly the screen examination as three-fourths of these cases were not mine for general diagnosis but were sent to me for screen examination. I also wish to emphasize the fact that it is a fluoroscopic, a screen examination, which helps. To take plates for the diagnosis of stomach conditions is like trying to diagnose lameness from a photograph. If the man has a half leg missing you may be quite sure he is lame, but if he only limps slightly you need to see him walk or see him on the movies to make sure that he is lame. Now the screen is our moving picture and we need to recognize defects in the movement of the stomach and intestines in order to make our diagnoses correctly. I can illustrate this best with the diagnosis of duodenal ulcer of the lower part of the organ, that is to say lower down than the entrance of the common duct. Six times cases have come to me with the clinical diagnosis of carcinoma, or pylorostenosis resulting from ulcer, with the classical syndrome of vomiting, loss of weight, pain after eating, etc. None of these six cases showed radiological signs of pylorostenosis nor of carcinoma but they did show the so-called reverse peristalsis of the duodenum, a moving back of the duodenal contents toward the stomach after forward progression had taken place. This may occur but once or constantly, according as the stenosis be mildly spastic or definitely organic. Once it is seen we know that there is some irritation at the lower end of the duodenum causing a spasm and this is an ulcer in ninety per cent. of the cases which I have seen. In the more severe cases a definite organic stenosis is produced which may be due to ulcer and its consequences or to a congenital band in the neighborhood of the ligament of Treitz. Two of my operated cases (Nos. 19 and 30) showed the syndrome of duodenal stenosis (Holzknecht):

(1) Filling of the duodenum above the seat of stenosis so that the wall of the duodenum is sharply outlined.

(2) Ineffectual or partly effectual peristalsis of the filled duodenum causing a change in the form of the organ but no change in the position of the contents. This is the so-called antiperistalsis.

(3) Retention above the stenosis.

(4) Occurrence of paralytic dilatation above the stenosis.

Only severe cases show the whole group of symptoms. The great majority show only the filling of the duodenum and the so-called antiperistalsis. The first of my two operative cases (No. 19) showed the filling and reversed motion of contents only during the first examination and at no subsequent examination. The second case (No. 30) showed only four or five waves of reversed motion and that after being induced by a special technic of my own. Incidentally I wish to mention that both of these cases showed old healed gastric ulcers. For this technic see Fortschr. a. d. Geb. d. Röntgen S. trahlen, April, 1914. It also appeared in Surg., Gynec. and Obst. (Dec., 1914). See also München. med. Wchnschr., Sept. 29, 1914, J. A. M. A., March 21, 1914.

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Case 30 presents an easy transition into another group of cases, the cases presenting a niche, a hole in the wall of the stomach. A true niche is usually found on the opposite side of the stomach. See Cases No. 15 and No. 37.

If you be not familiar with the term niche, let me explain that it is a hole in the wall of the stomach due to a callous penetrating ulcer which fills with bismuth and stays filled. I require three factors to make a diagnosis, first a bismuth filled hole projecting from the stomach shadow; secondly, the bismuth in this hole cannot be moved; thirdly, a pain point coinciding with the niche; fourthly, a gas bubble may be present above the projecting bismuth. I could move the bismuth out of the pseudoniche. As a matter of fact if I do not see a niche once in eighty to one hundred cases sent to me for examination I feel that I am not doing first-class screen work. In Vienna I used to see five or six a month out of their material, in Heidelberg, Halle and Breslau perhaps one in a month. This averaged up about one in eighty or a hundred cases. Seeing these holes in the wall of the stomach is a matter of technic.

Another point which I wish to bring out is that it is impossible in these cases to make any other diagnosis than penetrating ulcer with the possibility of ulcer carcinoma. I go into the subject of the niche so carefully because after making a diagnosis of penetrating ulcer two months ago one of the foremost surgeons of the city said to me, "You don't really see the penetrating ulcer, you infer it from the hour glass stomach." I merely wish to add that Case 11 presented a carcinoma engrafted on an older ulcer which I should undoubtedly have seen as a niche years previously. This presents the old story, the carcinoma patient comes too late to the diagnostic specialist and to the surgeon. In the six carcinoma patients which came to section I was able to make the direct positive localizing diagnosis in four (Cases Nos. 1, 2, 21 and 26); in one case (No. 4), I was able to make a diagnosis of a deforming lesion about the pylorus but was unable to say that it was carcinoma. In the sixth case I merely ruled out the clinical diagnosis of carcinoma of the sigmoid flexure but didn't remark anything about the stomach other than there was a disturbance in antrum peristalsis which might just as well have been due to adhesions from the palpable tumor. Really early diagnosis of carcinoma is just as impossible from the screen examination as it is with other methods. Many times, however, the radiological is the earliest diagnosis, particularly in the cases without palpable tumor. This is particularly true in scirrhus carcinoma (Case No. 2). This offers no great amount of satisfaction to the diagnostician as I have never seen an operable scirrhus. (By scirrhus I mean the tumor type which infiltrates and shrinks the wall of the stomach, not just any hard tumor.) Case 21 is very interesting from a diagnostic standpoint. The surgeon had made a diagnosis of cancer of the stomach, the radiological examination showed a probable carcinoma of the cardiac end of the stomach with marked stenosis of the esophagus. This

screen picture was shown to another medical consultant who later passed esophageal bougies whereafter the patient could eat solid food. He told me that the woman had cardiospasm. Gentlemen, cardiospasm with the ragged picture which this case showed is unknown to me. Cardiospasm cases show a smooth even dilatation. This patient was operated on two months later for a tumor in the abdomen and a carcinoma of the cardiac end of the stomach with general carcinomatosis was found. Cardiospasm with inanition is a very excellent diagnosis to leave alone until every organic lesion has been ruled out. Autopsy usually shows a lesion whether luetic, cancerous, or tubercular.

In my forty cases I have made but one bad positive mistake—No. 17. In a case of pancreatic tumor I saw besides the extraventricular tumor a small stomach almost hour glass in shape owing to smooth indentations on both sides. Peristalsis did not pass over this ring. I had previously only seen this condition in scirrhus carcinoma of the stomach. At operation the surgeon found a ring about the stomach which he called congenital. It did not disappear under anesthesia. I can find nothing similar described in the literature. I also declared a *spastic* hour glass stomach with penetrating ulcer to be an organic hour glass stomach (Case 37) and located a duodenal ulcer on the wrong side of the pylorus (Case 24). Considering that in these forty cases I have made 23 reports on the upper part of the duodenum, 17 negative and six positive in this operative series, I do not believe that the location of the ulcer on the wrong side of the pylorus is a grave offense. Incidentally I did the case without following my regular routine. Lately in these cases with six hour residue and some distortion of the duodenal bulb or cap (the first portion of the duodenum) I merely make a diagnosis of peri-pyloric ulcer, either gastric ulcer with adhesions or duodenal ulcer with a mild grade of pylorostenosis. In regard to a six-hour residue I have never seen a patient prepared properly who did not show either gastric ulcer or pylorostenosis if the residue were of considerable size and the proper meal had been given. The converse is not true.

Now I come to the last point which I shall take up this evening. Cole claims the first portion of the duodenum (the bulb of Holzkecht), the cap of Cole) is always without defect if there be no surgical duodenal ulcer present and no adhesions about the cap. I do not find this true as he claims in 100% of the cases but I certainly agree with Brewer's report of cases which Cole did for him. Brewer found that Cole was correct in 89% of the cases. Cole takes at least 36 plates to make a diagnosis. This is impracticable here as the expense is great. I accomplish nearly the same results on the screen with certain tricks (Bucky effect) and have had success in about the same proportion of cases. I believe that it is of value therefore in about 85% of all cases. Note Case No. 28 particularly where the bulb appeared like a button stuck on the end of the stomach instead of one of the normal shape.

Before I close I wish to call your attention to

Case No. 31, a subphrenic abscess, probably from a perforated duodenal ulcer in which many valuable days were lost in percussing and discussing, in passing stomach tubes and doing blood tests. The screen showed the condition at a flash. High up in the pleural cavity was the diaphragm, beneath that an air bubble about the size of two fists, and beneath that a fluid level which showed waves and splashes as the man was moved about. This merely clinches my belief that the strongest factor in the diagnosis of chronic gastrointestinal conditions is firstly, the history and secondly, the screen examination.

Gastric ulcer	
Penetrating ulcer.....	2
Simple ulcer.....	1
Duodenal ulcer	
Pars superior.....	2
Pars inferior.....	2
Pylorostenosis	
After ulcer.....	4
From adhesions.....	1
Carcinoma ventriculi	
Cardiac, involving esophagus.....	1
Other parts of stomach—	
Operable	1
Inoperable	5
Carcinoma esophagi.....	1
Tubercular salpinx with adhesions about sigmoid	1
Gallbladder adhesions to stomach.....	3
Adhesions about duod.....	1
Abscess probably arising from iliac perforation..	1
Stone in cystic duct.....	1
Inflammatory mass attached to spleen.....	1
Pancreatic tumor.....	1
Coloptosis	1
Visceroptosis	1
Adhesions about right dome of diaphragm.....	1
Gastroenterostomy	1
Chronic appendix.....	2
Subdiaphragmatic abscess.....	1
Adhesions of cecum to belly wall.....	1
Hypernephroma (growth above kidney) and gallstones	1
Retroperitoneal sarcoma (testicle).....	1
Abscess of liver.....	1

Case 1. Clin. diag. or question: Carcinoma ventriculi? probably not at pylorus. Clinical points: Pain in abdomen 14 months. No obstructive symptoms. No vomiting, except three times in month previous to operation. Lost 55 lbs. in one year. No palpable tumor. Radiological diagnosis: Inoperable carcinoma of pyloric end of stomach adherent to neighboring organs. Radiological points: Defect in stomach outline. Operative diagnosis: Inoperable carcinoma of stomach (pyloric end) grown into pancreas, adherent to liver.

Case 2. Clin. diag. or question: Carcinoma in abdomen, point of origin? Clinical points: Loss of weight followed by ascites with enlarged liver and palpable tumor in region of splenic flexure. Radiological diagnosis. Inoperable scirrhus carcinoma of stomach with adhesions. Radiological points: Contracted stomach with smooth irregular outline. Operative diagnosis: Inoperable scirrhus carcinoma of stomach. Pylorus and cardia free. Large cauliflower growth on back wall of stomach.

Case 3. Clin. Diag. or question: Tubercular ulcer of sigmoid. Clinical points: Virgin, difficult to examine, pain and rigidity in sigmoid region. Guaiac ++ in stool. Radiological diagnosis: Adhesions about sigmoid flexure, no gastric or duodenal ulcer, probably no tuberculous ulcer of colon. Radiological points: Colon filled in entirety absence of Stierlin's sign (absence of bismuth in colon about tuberculous ulcer), bulbus perfect.

Operative diagnosis: Tubercular salpinx adherent to sigmoid flexure, no ulcer of sigmoid.

Case 4. Clinical diag. or question: Ulcer or carcinoma of stomach? Gastric ptosis. Clinical points: Chronic indigestion for years with recent great loss of weight, no gastric or stool examinations were made, no palpable tumor. Radiological diagnosis: Probable old ulcer at pylorus with adhesions, no duodenal ulcer. (NOTE. Correct wording of radiological diagnosis should have been deforming lesion near pylorus, ulcer or carcinoma?). Radiological points: Enormous bulbus (associated most often with achylia), notch near pylorus constant during two days, bulbus perfect, stiffness of stomach wall on pressing in with finger. Operative diagnosis: Beginning carcinoma of lesser curvature of stomach with much involvement of glands. Little of stomach.

Case 5. Clinical diagnosis or question: Cholelithiasis, possibility of duodenal ulcer. Clinical points: Cholelithiasis history, pain in right hypochondrium, vomiting. Radiological diagnosis: Adhesions about pylorus, duodenum and gall bladder. No radiological signs of gastric ulcer. No duodenal ulcer. Radiological points: Stomach pulled markedly to right. Operative diagnosis: Adhesions at the pylorus and in gall bladder region. No cholelithiasis. No duodenal or gastric ulcer.

Case 6. Clinical diagnosis or question: Pyloric stenosis. Clinical points: Vomiting for eight years. No blood in vomitus. Residue in stomach. Radiological diagnosis: Pyloric stenosis. Adhesions about pylorus, duodenum, and gall bladder. Radiological points: Complete retention of bismuth eight hours after examination. Residue half-moon shaped. Operative diagnosis: Pyloric stenosis due to old ulcer. Adhesions about pylorus, duodenum and gall bladder.

Case 7. Clinical diagnosis: Carcinoma of splenic flexure. Possible abdominal aneurysm. Clinical points: Chronic indigestion. Palpable tumor. No stomach examination had been made. Radiological diagnosis: Abdominal tumor not connected with splenic flexure. Long so-called ptotic stomach. Radiological points: Palpable tumor at level of stomach bubble to right of splenic flexure. Disturbances in peristalsis near pylorus. Operative diagnosis: Carcinoma of lesser curvature of stomach chiefly involving glands. No tumor or splenic flexure.

Case 8. Clinical diagnosis or question: Cholelithiasis. Stenosis of small gut? Clinical points: No jaundice. Vomiting. Abdominal pain. Not localized in any particular spot. Appendix had been removed. Radiological diagnosis: Adhesions about gall bladder and pyloric region. No radiological signs of stenosis or gastric ulcer. No duodenal ulcer. Radiological points: Stomach pulled markedly to right. Operative diagnosis: Adhesions about pylorus and gall bladder region. No cholelithiasis. No gastric or duodenal ulcer.

Case 9. Clinical diagnosis or question: Gastric or duodenal ulcer? Clinical points: Vague abdominal and gastric disturbances since childhood. Radiological diagnosis: No radiological signs of gastric or duodenal ulcer. Probable adhesion of duodenum to belly wall. Radiological points: Constant notch about size of walnut on greater curvature of stomach. (Due to pressure of gas distended colon.) Operative diagnosis: Old cicatrix? at pylorus. Adhesions about duodenum. No duodenal or active gastric ulcer.

Case 10. Clinical diagnosis or question: Pylorostenosis. Malignancy? Clinical points. Duodenal ulcer perforation one year ago. Coffee ground vomitus for last few days. Marked loss of weight in past year. Radiological diagnosis: Marked gastrectasis, pyloric stenosis with adhesions about pyloric, duodenal and gall bladder regions. Radiological points: Almost complete retention of

bismuth six hours after meal. Halfmoon-shaped residue. Peristalsis active, at first, gradually disappearing. Operative diagnosis: Enormously dilated stomach, pylorostenosis. Adhesions in and about pyloric, duodenal and gall regions. No malignancy.

Case 11. Clinical diagnosis: Gastric ulcer. Malignancy? Clinical points: Vomiting. Loss of weight in last months. Old ulcer history. Radiological diagnosis: Fungus carcinoma of pyloric end of stomach adherent to neighboring organs. Radiological points: Defect in stomach shadow. Operative diagnosis: Cancer of pylorus arising from old callous ulcer which had at some previous time perforated into liver.

Case 12. Clinical diagnosis or question: Malignant tumor in stomach. Origin? Clinical points: Rapidly growing tumor in left part of abdomen: 60 lbs. loss of weight in three months. Radiological diagnosis: Radiologically negative stomach and duodenum. No constriction of large gut. Tumor probably not arising from colon. Radiological points: Fluid levels. Operative diagnosis: Abscess arising probably from small intestine infiltrating but not ulcerating colon. Diagnosis made after two operations.

Case 13. Clinical diagnosis or question: Duodenal ulcer? Malignancy? Clinical points: Ulcer history. Loss of weight. Radiological diagnosis: Ptosis and dilatation of stomach. Pylorostenosis with adhesions about pylorus. Duodenal ulcer? Radiological points: Almost complete retention of bismuth six hours after meal. Operative diagnosis: Marked pylorostenosis due to adhesions about duodenum and pylorus. Pancreas involved in mass. Probable duodenal ulcer. ? of malignancy.

Case 14. Clinical question or diagnosis: Stone in cystic duct? Pylorostenosis? Clinical points: Vomiting lasting for weeks without being able to retain any kind of food. Once jaundiced. Radiological diagnosis: No radiological signs of gastric ulcer, pyloro-stenosis, or carcinoma. Operative diagnosis: Atrophied gall-bladder. Stone in common duct. No pylorostenosis.

Case 15. Clinical diagnosis or question: Gastric ulcer. Pylorostenosis? Malignancy? Clinical points: Two and a half years' pain, relieved by vomiting. Aggravation of symptoms in last months. No hematemesis. Radiological diagnosis: Hour glass stomach (organic). Callous penetrating ulcer of upper sack. No duodenal ulcer. Radiological points: Haudek's niche with air bubble. Two separate sacks. Operative diagnosis: Callous ulcer of lesser curvature in organic upper sack of hour glass stomach. No duodenal ulcer.

Case 16. Clinical diagnosis or question: Gallstones. Clinical points: Tenderness in right hypochondrium on pressure. Vague distress to left of navel. Radiological diagnosis: No duodenal ulcer. No radiological signs of gastric ulcer or neoplasm. Possible tumor in region of left kidney. Radiological points: An inverted V-position of colon in erect posture with text book position of colon in supine position. Operative diagnosis: No ulcer of duodenum or stomach. Attached to spleen is a tumor mass of omentum about size of fist. Inflammatory in nature? No gallstones.

Case 17. Clinical diagnosis or question: Pancreatic tumor. Clinical points: Diabetes and tumor in abdomen. Loss of weight. Total HCl, 8. Radiological diagnosis: Palpable tumor not arising from stomach or duodenum. Scirrhus carcinoma of stomach. No duodenal ulcer. Radiological points: Small contracted stomach with constant narrowing about one and a half inches above pylorus. No peristalsis was noted above this ring. Operative diagnosis: Pancreatic cyst about size of tumor. No carcinoma but a ring about one and a half inches above narrowing of stomach which was present during the entire operation. No duodenal ulcer.

Case 18. Clinical diagnosis or question: Chronic appendicitis? Clinical points: Vague abdominal distress. Diarrhea. Radiological diagnosis: Enteroptosis. No radiological sign of chronic appendix. No gastric or duodenal ulcer. Operative diagnosis: Marked ptosis of stomach. No gastric or duodenal ulcer. Chronic appendix completely bound down.

Case 19. Clinical diagnosis or question: Ulcer location? Malignancy? Clinical points: Seven years of history of pain at varying times after eating. No vomiting until recently. Tarry stools. Rapid loss of weight. Radiological diagnosis: Stenosis of pars inferior duodeni. Stenosis has large spastic component. Ulcus duodeni? Radiological points: Duodenum shows peristaltic waves without advance of contents. Operative diagnosis: Ulcer of pars inferior duodeni. Old healed ulcer of greater curvature of stomach.

Case 20. Clinical diagnosis or question: No real clinical diagnosis. Possible duodenal ulcer. Clinical points: Vague abdominal distress. Some loss of weight. Vomits every morning since six years. Radiological diagnosis: No duodenal ulcer. No radiological signs of gastric ulcer or neoplasm. Isolated coloptosis atony of esophagus. Radiological points: Broad band of bismuth throughout esophagus remaining 30-40 seconds. Operative diagnosis: No gastric or duodenal ulcer. No gallstones. Appendix freely movable. Coloptosis.

Case 21. Clinical diagnosis or question: (Surgeon) Gastric carcinoma. (Medical Consultant) even after radiological examination, cardiospasm. Clinical points: One year vague digestive disturbances. Sensation of food sticking. Rapid loss of weight. Vomited solid food for months. After sounding by consultant could eat solid food. Two months later abdominal tumor. Radiological diagnosis: Stenosis of esophagus. Carcinoma of cardiac end (?) of stomach involving esophagus. Radiological points: Fluid bismuth mixture halts in esophagus assuming funnel form. On horizontal fluoroscope (trochoscope) cardiac end of lesser curvature is ragged. Bulbus duodeni perfect. Operative diagnosis: Two months later. Carcinoma of cardiac end of stomach involving esophagus. General carcinomatosis of abdomen including metastasis in ovary.

Case 22. Clinical diagnosis or question: Carcinoma of esophagus. Clinical points: Unable to swallow solid foods. Sensation of food sticking for some months. Radiological diagnosis: Stenosis of esophagus. Carcinoma? Radiological points: Bismuth halts two inches above esophagus for ten minutes. Operative diagnosis: Carcinoma of esophagus.

Case 23. Clinical diagnosis or question: Before first operation—appendix. Before second operation, two months later—subphrenic abscess. Clinical points: Mass in region of appendix. Temp. 103°. Pulse 140. White count 24,000. Septic temp. Radiological diagnosis. After first operation. No radiological signs of gastric or duodenal ulcer nor of neoplasm. No positive radiological diagnosis possible. Radiological points: After first operation. Fixation of right dome of diaphragm seemed to make clinical diagnosis stronger. Operative diagnosis: First operation. Appendix not cause of trouble. No focus of pus found. Mass was omentum. Second operation. Nothing found except adhesions between liver and diaphragm.

Case 24. Clinical diagnosis or question: Ulcer. Where? Clinical points: Hematemesis, six days before. Ulcer history. Pylorostenosis had not been ruled out clinically. Radiological diagnosis: Ulcer (probably gastric). No duodenal ulcer. Real diagnosis should have been peripyloric ulcer. Radiological points: Bulbus duodeni well filled out. (Done out of routine.) Six hour residue. Radiological diagnosis: Duodenal ulcer. Mild grade of pylorostenosis.

Case 25. Clinical diagnosis or question: Previous to radiological examination. Adhesions. Good functioning gastroenterostomy? Clinical points: Patient complains of pain but no vomiting at present. Operated three months ago for gastric ulcer. Radiological diagnosis: Good functioning gastroenterostomy wound. Pyloric end of stomach blocked off. Radiological points: No bismuth seen to enter duodenum. Operative diagnosis: Informed afterwards that pyloroeotomy had been performed.

Case 26. Clinical diagnosis or question: Carcinoma ventriculi. Clinical points: Large tumor mass in right hypogastrium since three months tumor or mass about size of walnut in epigastrium, movable. Much vomiting in last three months until one week ago; 22 lbs. loss in weight. Radiological diagnosis: Tumor outside of stomach continuous with liver shadow. Infiltration of pylorus. Carcinoma ventriculi. Radiological points: Stomach normal in shape. Movable tumor coincides with pylorus on palpation and moves with it. Antrum peristalsis defective. Slight irregularity of lesser curvature of stomach, one-sixth residue in stomach despite good peristalsis.

Case 27. Clinical diagnosis or question: Pylorostenosis? Clinical points: 23 years old, 25 lbs. loss of weight in last few months. Operated on for appendicitis to cure stomach two and one-half years ago. Vomited after meals off and on for last years. For nine years pain in epigastrium in afternoons and at 11 P. M. Eating made pains worse. Radiological diagnosis: Pylorostenosis. Old peripyloric ulcer, probably duodenal in origin. Adhesions in pyloric, duodenal, and gallbladder region. Radiological points: Large dilated stomach drawn to right. "Stenosen" peristalsis, i. e., violent peristalsis. After 24 hours one-third bismuth residue in stomach. Operative diagnosis: Pylorostenosis. Dense mass of adhesions involving gallbladder and duodenum to lesser curvature of stomach.

Case 28. Clinical diagnosis or question: Peptic ulcer. Radiological diagnosis: No radiological signs of gastric ulcer. Hypersecretion. Duodenal ulcer? Radiological points: No residue in stomach. Button bulbus. Fasting stomach filled with fluid (steerhorn stomach). Operative diagnosis: Duodenal ulcer.

Case 29. Clinical diagnosis or question: Chronic appendix. Duodenal ulcer? Clinical points: Tenderness in right iliac region. Vague gastric symptoms. Radiological diagnosis: Chronic appendix hanging from lower end of coecum? Radiological points: Bulbus perfect. Operative diagnosis: Chronic appendix not retrocecal.

Case 30. Clinical diagnosis or question: Ulcer? Carcinoma? Clinical points: 14 years ago stomach pleated for gastric ulcer. Six months ago began to lose weight. No vomiting. Appetite poor. Radiological diagnosis: Hourglass stomach, probably spastic. Spasmodic stenosis of duodenum in neighborhood of ligament of Treitz. Probable old gastric ulcer with more recent ulcer of the lower part of duodenum. No duodenal ulcer of upper part. Adhesions. Radiological points: Pseudo-niche on posterior wall of stomach. Knife-like indrawing of stomach, dividing it into two parts. Reversed motion of duodenal contents (so-called antiperistalsis). Operative diagnosis: Ulcer of lower part of duodenum. Old gastric ulcer (thickening at pylorus).

Case 31. Clinical diagnosis or question: Inflammatory condition of abdomen. Carcinoma? Clinical points: Six months ago gas formation, six weeks ago stomach specialist. No dark stools. No vomiting. Four weeks ago severe attack of abdominal with "hard muscles" after lobster dinner. No fever. In bed one week. One week ago

no free HCl. Ascites. Swelling of legs. Radiological diagnosis: Gas containing subphrenic abscess on right. Adhesions about duodenum, distortion of duodenum and large bowel. Duodenal ulcer? Radiological points: Beneath diaphragm a fluid level which shows waves on moving patient. Duodenum distorted lying along base of liver shadow moving with it. Liver indents upper part of stomach. Operative diagnosis: Subdiaphragmatic abscess. Large amount of stinking pus exudes from wound.

Case 32. Clinical diagnosis or question: Adhesions. ? cause of vomiting. Neoplasm? Clinical points: Six weeks ago ectopic pregnancy? Removal of tumor and portion of gut. Radiological diagnosis: Pylorostenosis. Infiltration of pylorus. Carcinoma ventriculi? Fluid levels probably indicate obstruction to bowel. Radiological points: Disturbance of antrum peristalsis which is violent. Fluid levels in bowels (constant); four-fifths bismuth residue after six hours. Operative diagnosis: Adhesions which kinked gut in places. Pylorus blocked by adhesions. No carcinoma.

Case 33. Clinical diagnosis or question: Ulcer? Clinical points: Two years ago gas pains. No vomiting. Pain on right just below navel. Lost 10-15 lbs. weight. Radiological diagnosis: Duodenal ulcer with beginning pylorostenosis or gastric ulcer at pylorus. (Peripyloric ulcer.) Radiological points: Six hours residue. Operative diagnosis: Gastric ulcer at pylorus.

Case 34. Clinical diagnosis or question: Ulcer? Adhesions? Clinical points: One year ago appendectomy. Pain has no relation to eating. Radiological diagnosis: No radiological sign of gastric ulcer. Probable adhesions about stomach, cecum, proximal part of transverse colon. Chronic constipation of ascendens type. Radiological points: Stomach and colon transversum only fairly movable. Operative diagnosis: Adhesions from cecum to belly wall. Jackson's membrane about colon.

Case 35. Clinical diagnosis or question: Abscess of liver. Amebiasis? Carcinoma ventriculi? Clinical points: One year diarrhea with mucus, blood, pus. Rapidly growing tumor in right hypochondrium for last few days. Radiological diagnosis: Tumor probably connected with liver. Radiological points: Stomach pressed far to left. Indentation by liver. Antrum formation of stomach distorted. Operative diagnosis: Liver abscess.

Case 36. Clinical diagnosis or question: Carcinoma of stomach? Gallstones. Malaria? After stool and radiological examination—malignancy? Adrenal disease? Clinical points: Gallstone attacks in previous years. Nausea, retching, belching after meals, six months' duration; 50 lbs. loss in last six months a/c daily diarrhea. Asthenia. Temperature 102° every second night. Negative malarial parasites. No occult blood. Stool (Schmidt test diet) type of early toxic diarrhea. No abnormal elements. B. P. 150 mm. Radiological diagnosis: No radiological signs of carcinoma of stomach. Tuberculosis of right apex. Radiological points: Rigidity of upper part of stomach. Operative diagnosis: Gallstones. Hypernephroma.

Case 37. Clinical diagnosis or question: Ulcer? Clinical points: Twenty-two years suffered off and on with gas pains after meals. Worse for last six months. Occasional vomiting. Radiological diagnosis: Callous penetrating ulcer of stomach. Organic hour-glass stomach. Radiological points: Niche (hole in the wall of stomach). Lower sack of stomach takes 25 minutes to fill on two successive days. Operative diagnosis: Ulcer penetrating into pancreas. Large dilated stomach with infolding but no hour-glass stomach.

Case 38. Clinical diagnosis or question: Vis-

ceroptosis. Clinical points: Appendix operation two and a half years ago. After operation vomiting. Since then miserable. Radiological diagnosis: No radiological signs of gastric or duodenal ulcer nor of any stenosis. Constipation of ascendens type. Radiological points: Bulbus duodeni perfect. Operative diagnosis: No gastric or duodenal ulcer. Visceroptosis.

Case 39. Clinical diagnosis or question: Appendix? Ulcer? Carcinoma? Clinical points: Five years ago severe pain in abdomen. Since one year severe pain in epigastrium right after eating. Nausea in mornings. No pain if he doesn't eat. Never vomited. Lost 20 lbs. in last four weeks. Radiological diagnosis: No radiological signs of active gastric ulcer or neoplasm. Probable chronic appendix (retrocecal?). Radiological points: Point of maximum pain coincides with cecum. Operative diagnosis: Old healed ulcer of stomach connected by band of adhesions with gallbladder. Chronic retrocecal appendix.

Case 40. Clinical diagnosis or question: Gastric ulcer with perforation? Carcinoma ventriculi? After radiological examination—gastric ulcer with perforation? Retroperitoneal sarcoma with metastases. Clinical points: Acute pain in left hypogastrium for several months. Pain constant. Two months ago testicle removed. No blood in stools. Age 27. Rapidly growing tumor in left hypogastrium for several days. Radiological diagnosis: Large dilated stomach. Extraventricular tumor. Radiological points: Pseudo-filling defect. Operative diagnosis: Retroperitoneal sarcomatous metastases (cystic and solid).

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Discussion.

Dr. W. C. Alvarez: There is little to add to this interesting paper. I am only afraid that in confining himself so closely to this phase of the subject, Dr. Lippman may seem to have made the subject clearer than it really is. I think that those who do this work find that the average case is not as clear and as beautiful as these that we have had described to us tonight. So many show either nothing definite or functional disturbances which may or may not have pathological significance. Dr. Lippman's material may be unusual in that it is largely referred to him by surgeons who would be more likely to get severe, long-standing surgical cases. Statistics compiled by Fenwick, Friedenwald and others show that about 85% of patients with gastrointestinal disease have functional disturbances. Granting that every year better diagnostic methods transfer a number of cases to the organic column, still, I think even in consultant practice, we will find eight or nine doubtful or normal cases to one in which there is a beautiful ulcer or carcinoma defect, hour-glass stomach, etc. Even in the ulcer cases I believe we will oftener find disturbances of function than craters and signs of perforation.

A word as to six hour stasis as diagnostic of organic lesions at the pylorus. I frequently find one or two ounces of bismuth in the stomach six hours after a meal when no pyloric lesion can be found. This may be present on occasions and absent on others. Very interesting is the fact that the stomach will empty rapidly in the first forty-five minutes and then there will be practically no change in the remainder for six to eight hours. In these cases I believe the trouble is to be found in the intestine. I saw recently a case in which a half ounce of bismuth remained in the stomach twenty-three hours. There was no sign of ulcer or carcinoma and the other findings were against

such a diagnosis. Besides, she had had stasis symptoms for five years. Fig-skins were vomited eight days after she had eaten them. What she had was a markedly prolapsed uterus which I believe can explain the findings.

Dr. Lippman, closing discussion: My material is divided into consultation work chiefly from one group of surgeons and several groups of medical men—I average about 72% nonoperative and 28% operative cases. Dr. Alvarez mentions one or two ounces of bismuth residue after six hours—I only use one and one-third ounces for a meal and this would constitute a complete retention. I wish to reiterate I have never seen complete retention nor a large (I emphasize the word large) residue without organic lesion of some kind. The case which Dr. Alvarez quotes is unfortunately not an operated case. I confined my talk to operatively confirmed diagnoses. I think with the history which he offers and the findings that he might have found as I have in several similar cases reversed motion of the duodenal contents (so-called antiperistalsis) and duodenal ulcer of the lower part of the duodenum.

GENERAL PARESIS AND ITS RELATION TO SYPHILIS, WITH A REPORT OF THE PATHOLOGIST OF NAPA STATE HOSPITAL.

By A. W. HOISHOLT, M. D., Medical Superintendent, Napa State Hospital; Clin. Prof. Psychiatry, Stanford University.

General paresis, general paralysis of the insane or softening of the brain was first spoken of by Willis in 1672, but was not recognized and described as a disease entity until 1822, when the French alienist, Bayle, pronounced the somatic and psychic symptoms manifestations of one and the same disease. From 1822 to the middle of the last century, the efforts of investigators were centered on clinically outlining the disease-picture. At the end of the fifties, observations began to be made of the frequent occurrence of syphilitic infection in the history of general paralytics. Esmarch and Jessen in 1857, Steenberg in 1860, and Jespersen in 1874 first drew attention to this relationship; the latter in an article entitled "*Is Progressive General Paresis Due to Syphilis?*" Careful statistic researches in this direction were later made by Westphal, Erb, Fournier and Krafft-Ebing, and in the course of years, the percentage of general paresis, showing a history of probable syphilitic infection, gradually rose until it reached 85 to 90%, and in the cases of juvenile paresis, even higher. The medical profession was therefore quite ready to accept the results which were established by the Wassermann and Noguchi reactions, the Nonne—Phase I reaction of globulin increase, and the increase of the lymphocytes in the cerebrospinal fluid. Since these sero-diagnostic tests (known also as "the four reactions") have been recognized as indicators of luetic nerve degeneration, the assertion that "Without a previous syphilitic infection, there can be no general paresis" has become generally accepted.

The question was finally definitely decided when Noguchi a couple of years ago succeeded in demonstrating the finding of treponema pallidum in the brain in about 25 per cent. of 200 cases of general paralysis and especially when he, over a year ago, was able to produce typical syphilitic